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Programmed cell death during regression of the MCF-7 human breast cancer following estrogen ablation.

Kyprianou N, English HF, Davidson NE, Isaacs JT.

Cancer Res. 1991 Jan 1;51(1):162-6.

Johns Hopkins Oncology Center, Baltimore, Maryland 21205.

To study the mechanism of regression of human mammary cancer following estrogen ablation, estrogen-responsive MCF-7 human mammary adenocarcinoma cells were inoculated into ovariectomized female nude mice supplemented with exogenous 17 beta-estradiol (E2) via an E2 implant. Implants were then removed when MCF-7 tumors were 400 mm3 in size. Removal of the E2 implants resulted in a 50% tumor regression by 2 weeks following E2 ablation. Associated with this regression is a rapid (i.e., within 1 day following E2 ablation) enhanced expression of the transforming growth factor beta 1 and TRPM-2-genes, two genes the expression of which has been previously demonstrated to be enhanced in a variety of cell types induced to undergo programmed cell death (i.e., apoptosis). The enhanced expression of transforming growth factor beta 1 and TRPM-2 is not a nonspecific response since the expression of other genes, like c-fos, c-H-ras, and pS2, decrease following E2 ablation. Fragmentation of tumor DNA into nucleosomal oligomers and histological appearance of apoptotic bodies are characteristic early events that precede the dramatic reduction in tumor volume following E2 ablation. These results demonstrate that the regression of MCF-7 human mammary cancers in nude mice following estrogen ablation is due to a sequence of biochemical and morphological changes that result in both the cessation of cell proliferation and activation of programmed death or apoptosis of these MCF-7 cancer cells. Clarification of the biochemical pathway involved in the activation of this programmed cell death should identify new targets of therapy for even estrogen-independent

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Anti-HER2 antibody enhances the growth inhibitory effect of anti-oestrogen on breast cancer cells expressing both oestrogen receptors and HER2.

Kunisue H, Kurebayashi J, Otsuki T, Tang CK, Kurosumi M, Yamamoto S, Tanaka K, Doihara H, Shimizu N, Sonoo H.

Br J Cancer. 2000 Jan;82(1):46-51.

Department of Breast and Thyroid Surgery, Kawasaki Medical School, Okayama, Japan.

Anti-oestrogen is effective for the treatment of oestrogen receptor (ER)-positive breast carcinomas, but most of these tumours become resistant to anti-oestrogen. It has been suggested that anti-oestrogen therapy may induce a HER2 signalling pathway in breast cancer cells and this may cause resistance to anti-oestrogen. Thus, it is conceivable that combined therapy with anti-oestrogen and anti-HER2 antibody might be more effective. In the present study, we investigated the effect of combined treatment with a humanized anti-HER2 monoclonal antibody, rhumAbHER2 (trastuzumab), and an anti-oestrogen, ICI 182,780, on the cell growth of three human breast cancer cell lines which respectively express different levels of ER and HER2. The combined treatment enhanced the growth inhibitory effect on ML-20 cells, which express a high level of ER and a moderate level of HER2, but showed no additive effect on either KPL-4 cells, which express no ER and a moderate level of HER2, or MDA-MB-231 cells, which express no ER and a low level of HER2. It is also suggested that both the antibody and anti-oestrogen induce a G1-S blockade and apoptosis. These findings indicate that combined treatment with anti-HER2 antibody and anti-oestrogen may be useful for the treatment of patients with breast cancer expressing both ER and HER2.

Publication Types:
- Journal Article

Chemo-signal therapy, an emerging new approach to modify drug resistance in breast cancer.

Pusztai L, Esteva FJ, Cristofanilli M, Hung MC, Hortobagyi GN.

Cancer Treat Rev. 1999 Oct;25(5):271-7.

Department of Breast Medical Oncology, The University of Texas MD Anderson Cancer Center, 1515 Holcombe Blvd, Houston, TX 77030, USA.

Recent advances in understanding how response or resistance to cytotoxic drugs develops at the cellular level resulted in the development of novel, non-cytotoxic agents that modulate response the chemotherapy. 'Chemo-signal therapy', the combination of chemotherapy with cellular response modifiers, is a very promising new treatment modality that has entered the arena of clinical trials. Clinical experience with the anti-HER-2 antibody, trastuzumab, in breast cancer has demonstrated that manipulation of growth factor signalling can enhance sensitivity to cytotoxic drugs in a clinically meaningful way. Several other agents that were designed to modulate response to chemotherapy are currently in early phases of clinical drug development. It is likely that some of these new molecules will have a major impact on how chemotherapy will be given in the next decade. This paper will review current clinical research with a select group of chemotherapy response modifiers. We will focus on agents that modulate signal transduction, oncogene expression and apoptosis with an emphasis on breast cancer.

Publication Types:
- Journal Article
- Review
- Review, Tutorial